Alternative Application for Metallo-Organic Framework (MoF) for Arterial Plaque Removal via Solid-State Pump Mechanism Designed to Attach to Cholesterol-Laden Foam Cells

22 November 2022
Simon Edwards
Research Acceleration Initiative

Introduction

One of the most challenging problems in medicine today may not be curing cancer or slowing the aging process, but rather the deceptively simple problem of arterial plaque buildup. Congestive heart failure and other conditions such as ischemic stroke and aortic embolism and dissection all have their basis in the buildup of plaques. While there are many existing methods for dissolving or dislodging plaques, none of these are able to achieve those ends without fatally damaging the patient. Anti-phospholids attack all living cells, not only plaques. Ultrasound has been found effective for dislodging plaques, but the act of dislodging plaques frequently leads to ischemia. Any medically effective therapy resolving arterial plaques must therefore be specific to the plaques and must not lead to large chunks of plaque breaking off and lodging in smaller blood vessels.

Abstract

Metallo-Organic Frameworks, which have shown promise as a chemotherapy delivery mechanism, I would suggest, have alternative application for resolution of arterial plaques.

The best current evidence that this is possible is the fact that plasmacytoid dentritic cells (pDCs,) despite being responsible for the initial arterial wall corrosion that makes plaque formation possible, have been shown to help to attrite existing plaques, presumably through the secretion of type 1 interferons.

One would speculate that the induction of inflammation i.e. hyper-hydrolysation/overpressure within smooth muscle cells generally is capable of triggering them to release their contents. In order for foam cells, which are essentially hollowed out smooth muscle cells, to store cholesterol, they have to first eject their original contents. Healthy smooth muscle as found in arterial walls is flexible and expandable, an attribute that helps to stave off blockages and ruptures. It is this very flexibility that makes corrupted smooth muscle cells (known as foam cells) capable of storing large quantities of cholesterol. Since the proteins stored within the foam cells are already fully hydrolyzed, they are already at their natural limit in terms of carrying capacity of water. This would suggest that little additional fluid pressure (coming from the interior of the cells) would be needed to effectively disintegrate the cells.

Since the exterior of foam cells and the exterior of healthy smooth muscle is identical in terms of receptors, the proposed MoF must be specifically adhere to cells based upon spacing and angular orientation of receptors on the foam cell walls. Where the receptors on the walls of healthy smooth muscle should

be relatively close together and oriented in a mostly uniform direction, the cholesterol-laden foam cells are bloated with receptors spaced far apart and oriented at differing angles, like a balloon with spikes as opposed to a component of a smooth tract with cilia-like structures protruding like stubble.

The internal structure of the MoF would need to be highly hydrophilic and capable of acting as a solid-state pump, generating an overpressure of water in the foam cells once attached. To be clear, all this MoF structure would need to do is to attach to the foam cells and to pull plasma from the bloodstream and pump it into the foam cells, causing an excess of pressure that alters the internal behavior of the cell, ultimately leading to its disintegration.

The remaining healthy smooth muscle is not damaged by the hypothetical MoF treatment as the MoF's size and curved structure allows it only to attach to curved surfaces 22-28.5nm in diameter with the appropriate receptors. This means that the MoFs for this application would likely be a partial toroid (doughnaught-shape,) facilitating attachment to the curved surface of the foam cells.

After treatment, the MoFs would eventually degrade and be digested by the body, as would the corrupted foam cells. Beneath the corroded arterial wall cells would be a layer of healthy smooth muscle. This newly exposed smooth muscle would be of sufficient thickness to safely perform its function and the removal of the diseased inner surface layer of the artery would be the equivalent of exfoliating the outer layer of the dermis. Unlike the skin, the walls of the blood vessels would not stand up to repeated cycles of corrosion and corrosion removal. The ability of the arterial walls to withstand repeated cycles of plaque removal would be comparable to the ability of iron pots to withstand rust removal. One, or perhaps two cycles could be tolerated, but likely no more. Beyond this, the arterial walls would be thinned and rendered prone to embolism and subsequent dissection.

Conclusion

This treatment, although it would undo decades of plaque buildup, would not perform the task of preventing the buildup process from starting again. To achieve this, the plasmacytic dendritic cells would need to be reprogrammed so as to prevent them from attacking the arterial walls in addition to the plaque removal treatment.